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Term: l1 with l2

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DATE: Friday, May 14, 2004 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=PGPB,USPT; PLUR=YES; OP=AND			
L3	l1 with l2	10	L3
L2	(treat$ or protect$) near6 (neural adj tissue or brain or cns or central adj nervous adj system or cerebellum or cerebrum)	16502	L2
L1	(neuronal or neural) near3 progenitor adj cell	481	L1

END OF SEARCH HISTORY

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Search Results - Record(s) 1 through 10 of 10 returned.

☐ 1. 20040034049. 01 Apr 03. 19 Feb 04. Promoters for the proliferation and differentiation of stem cells and/or neuron precursor cells. Okawa, Shigenori, et al. 514/278; 514/409 A61K031/4747.

☐ 2. 20030223972. 14 Feb 03. 04 Dec 03. Myelination of congenitally dysmyelinated forebrains using oligodendrocyte progenitor cells. Goldman, Steven A., et al. 424/93.21; 435/368 435/456 435/458 435/459 A61K048/00 C12N005/08 C12N015/86 C12N015/88 C12N015/87.

☐ 3. 20030203844. 18 Sep 02. 30 Oct 03. Treatment of central nervous system disorders. Delfani, Kioumars, et al. 514/12; A61K038/18.

☐ 4. 20030082160. 24 Sep 02. 01 May 03. Differentiation of whole bone marrow. Yu, John S., et al. 424/93.21; 435/368 A61K048/00 C12N005/08.

☑ 5. 20030049234. 18 Mar 99. 13 Mar 03. DISCOVERY, LOCALIZATION, HARVEST, AND PROPAGATION OF AN FGF2 AND BDNF-RESPONSIVE POPULATION OF NEURAL AND NEURONAL PROGENITOR CELLS IN THE ADULT HUMAN FOREBRAIN. GOLDMAN, STEVEN A., et al. 424/93.21; 435/368 A61K048/00 C12N005/08.

☐ 6. 20030003090. 23 May 02. 02 Jan 03. Directed in vitro differentiation of marrow stromal cells into neural cell progenitors. Prockop, Darwin J., et al. 424/93.21; 435/368 A61K048/00 C12N005/08.

☐ 7. 20020037281. 25 May 01. 28 Mar 02. Methods of transducing neural cells using lentivirus vectors. Davidson, Beverly L., et al. 424/93.21; 435/368 435/456 C12N015/867 A61K048/00 C12N005/08.

☐ 8. 20010024827. 08 May 01. 27 Sep 01. Neuronal progenitor cells and uses thereof. Luskin, Marla B.. 435/375; 435/6 C12Q001/68 C12N005/08.

☐ 9. 6251669. 05 Jan 98; 26 Jun 01. Neuronal progenitor cells and uses thereof. Luskin; Marla B.. 435/375; 424/93.21 435/6 435/69.1. C12N005/00 C12N005/02 C12Q001/68 C12P021/06 A61K048/00.

☑ 10. 5753505. 06 Jul 95; 19 May 98. Neuronal progenitor cells and uses thereof. Luskin; Marla B.. 435/375; 424/93.21 435/6 435/69.1. C12N005/00 C12N015/09 A61K048/00.

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Terms	Documents
L1 with L2	10

h e b b g e e e f e e e e che

09/913,427

=> d his

(FILE 'HOME' ENTERED AT 15:02:07 ON 14 MAY 2004)

FILE 'MEDLINE, CAPLUS, BIOSIS, SCISEARCH' ENTERED AT 15:02:23 ON 14 MAY 2004

L1 3012 S (NEURONAL OR NEURAL)(3A)PROGENITOR(W)CELL
L2 66482 S (TREAT? OR PROTECT?)(6A)(NEURAL(W)TISSUE OR BRAIN OR CNS OR C
L3 73 S L1 AND L2
L4 13 S L1(9A)L2
L5 11 DUP REM L4 (2 DUPLICATES REMOVED)
L6 47 DUP REM L3 (26 DUPLICATES REMOVED)

=> d au ti so 21-47 l6

L6 ANSWER 21 OF 47 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
AU Chen J L; Li Y; Wang L; Lu M; Chopp M (Reprint)
TI Caspase inhibition by Z-VAD increases the survival of grafted bone marrow cells and improves functional outcome after MCAo in rats
SO JOURNAL OF THE NEUROLOGICAL SCIENCES, (15 JUL 2002) Vol. 199, No. 1-2, pp. 17-24.
Publisher: ELSEVIER SCIENCE BV, PO BOX 211, 1000 AE AMSTERDAM, NETHERLANDS.
ISSN: 0022-510X.

L6 ANSWER 22 OF 47 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AU Jarman, R. G. [Reprint Author]; Freed, C. R. [Reprint Author]
TI HUMAN **NEURAL PROGENITOR CELLS** SHOW NORMAL VENTROLATERAL MIGRATION IN ORGAN CULTURES OF HUMAN EMBRYONIC MESENCEPHALON.
SO Society for Neuroscience Abstract Viewer and Itinerary Planner, (2002) Vol. 2002, pp. Abstract No. 726.7. http://sfn.scholarone.com. cd-rom. Meeting Info.: 32nd Annual Meeting of the Society for Neuroscience. Orlando, Florida, USA. November 02-07, 2002. Society for Neuroscience.

L6 ANSWER 23 OF 47 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AU Stauffer, W. R. [Reprint Author]; Fellows-Mayle, W. [Reprint Author]; Niranjan, A. [Reprint Author]; Tyler-Kabara, E. [Reprint Author]; Hong, C. S.; Glorioso, J.; Gobbel, G. T. [Reprint Author]
TI BRAIN IRRADIATION ENHANCES SURVIVAL OF NEURAL PROGENITOR ALLOGRAFTS TRANSPLANTED INTO NORMAL AND TUMOR-BEARING BRAINS.
SO Society for Neuroscience Abstract Viewer and Itinerary Planner, (2002) Vol. 2002, pp. Abstract No. 237.14. http://sfn.scholarone.com. cd-rom. Meeting Info.: 32nd Annual Meeting of the Society for Neuroscience. Orlando, Florida, USA. November 02-07, 2002. Society for Neuroscience.

L6 ANSWER 24 OF 47 MEDLINE on STN
AU Okano H
TI Neural stem cells: the basic biology and prospects for brain repair.
SO Rinsho shinkeigaku. Clinical neurology, (2001 Dec) 41 (12) 1036-40.
Journal code: 0417466. ISSN: 0009-918X.

L6 ANSWER 25 OF 47 MEDLINE on STN DUPLICATE 5
AU Vaccarino F M; Ganat Y; Zhang Y; Zheng W
TI Stem cells in neurodevelopment and plasticity.
SO Neuropsychopharmacology : official publication of the American College of Neuropsychopharmacology, (2001 Dec) 25 (6) 805-15. Ref: 104
Journal code: 8904907. ISSN: 0893-133X.

L6 ANSWER 26 OF 47 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
AU Viktorov I V (Reprint)
TI Stem cells of mammalian brain: Biology of the stem cells in vivo and in vitro

SO BIOLOGY BULLETIN, (NOV-DEC 2001) Vol. 28, No. 6, pp. 544-552.
Publisher: MAIK NAUKA/INTERPERIODICA, C/O KLUWER ACADEMIC-PLENUM PUBLISHERS, 233 SPRING ST, NEW YORK, NY 10013-1578 USA.
ISSN: 1062-3590.

L6 ANSWER 27 OF 47 CAPLUS COPYRIGHT 2004 ACS on STN
AU Kitagawa, Kazuo; Matsumoto, Masayasu; Hori, Masatsugu
TI Protective and regenerative response endogenously induced in the ischemic brain
SO Canadian Journal of Physiology and Pharmacology (2001), 79(3), 262-265
CODEN: CJPPA3; ISSN: 0008-4212

L6 ANSWER 28 OF 47 CAPLUS COPYRIGHT 2004 ACS on STN DUPLICATE 6
AU Lu, Chengbiao; Fu, Weiming; Mattson, Mark P.
TI Telomerase protects developing neurons against DNA damage-induced cell death
SO Developmental Brain Research (2001), 131(1,2), 167-171
CODEN: DBRRDB; ISSN: 0165-3806

L6 ANSWER 29 OF 47 CAPLUS COPYRIGHT 2004 ACS on STN
AU Okano, Hideyuki; Sakakibara, Shin-ichi; Sawamoto, Kazunobu; Nakamura, Yuki; Kaneko, Yukiko; Akamatsu, Wado; Tokunaga, Akinori; Imai, Takao; Miyata, Takaki; Shimazaki, Takuya
TI Regulatory mechanisms for the differentiation of neural stem cells
SO International Congress Series (2001), 1222(Tissue Engineering for Therapeutic Use 5), 11-19
CODEN: EXMDA4; ISSN: 0531-5131

L6 ANSWER 30 OF 47 CAPLUS COPYRIGHT 2004 ACS on STN
IN Eriksson, Peter; Orwar, Owe
TI A method for introducing nucleic acids into **neural** stem or **progenitor cells** via the inherent transport system of the cell
SO PCT Int. Appl., 26 pp.
CODEN: PIXXD2

L6 ANSWER 31 OF 47 CAPLUS COPYRIGHT 2004 ACS on STN
IN Eriksson, Peter
TI Growth hormone-modulating agents and method for treatment of conditions affecting **neural** stem cells or **progenitor cells**
SO PCT Int. Appl., 22 pp.
CODEN: PIXXD2

L6 ANSWER 32 OF 47 CAPLUS COPYRIGHT 2004 ACS on STN
IN Snyder, Evan Y.; Lynch, William P.; Breakefield, Xandra O.; Aboody, Karen
TI Engraftable neural progenitor and stem cells for brain tumor therapy
SO PCT Int. Appl., 33 pp.
CODEN: PIXXD2

L6 ANSWER 33 OF 47 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AU Peterson, Daniel A. [Reprint author]; Ray, Jasodhara; Gage, Fred H.
TI Future prospects of gene therapy for **treating CNS** diseases.
SO Emerich, Dwaine F. [Editor]; Dean, Reginald L., III [Editor]; Sanberg, Paul R. [Editor]. (2000) pp. 485-508. Contemporary Neuroscience. Central nervous system diseases: Innovative animal models from lab to clinic. print.
Publisher: Humana Press Inc., 999 Riverview Drive, Suite 208, Totowa, NJ, 07512, USA.
ISBN: 0-89603-724-X (cloth).

L6 ANSWER 34 OF 47 MEDLINE on STN DUPLICATE 7
AU Benedetti S; Pirola B; Pollo B; Magrassi L; Bruzzone M G; Rigamonti D;

Galli R; Selleri S; Di Meco F; De Fraja C; Vescovi A; Cattaneo E; Finocchiaro G
TI Gene therapy of experimental brain tumors using **neural progenitor cells**.
SO Nature medicine, (2000 Apr) 6 (4) 447-50.
Journal code: 9502015. ISSN: 1078-8956.

L6 ANSWER 35 OF 47 MEDLINE on STN DUPLICATE 8
AU Kaneko Y; Sakakibara S; Imai T; Suzuki A; Nakamura Y; Sawamoto K; Ogawa Y; Toyama Y; Miyata T; Okano H
TI Musashi1: an evolutionally conserved marker for CNS progenitor cells including neural stem cells.
SO Developmental neuroscience, (2000) 22 (1-2) 139-53.
Journal code: 7809375. ISSN: 0378-5866.

L6 ANSWER 36 OF 47 MEDLINE on STN DUPLICATE 9
AU Okano H
TI Neural stem cells: the basic biology and prospects for brain repair.
SO Nihon shinkei seishin yakurigaku zasshi = Japanese journal of psychopharmacology, (2000 Feb) 20 (1) 21-6. Ref: 35
Journal code: 9509023. ISSN: 1340-2544.

L6 ANSWER 37 OF 47 CAPLUS COPYRIGHT 2004 ACS on STN
IN Reid, James Steven; Fallon, James H.
TI Methods for treating neurological deficits
SO PCT Int. Appl., 100 pp.
CODEN: PIXXD2

L6 ANSWER 38 OF 47 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
AU Alder J; Lee K J; Jessell T M; Hatten M E (Reprint)
TI Generation of cerebellar granule neurons in vivo by transplantation of BMP-treated **neural progenitor cells**
SO NATURE NEUROSCIENCE, (JUN 1999) Vol. 2, No. 6, pp. 535-540.
Publisher: NATURE AMERICA INC, 345 PARK AVE SOUTH, NEW YORK, NY 10010-1707.
ISSN: 1097-6256.

L6 ANSWER 39 OF 47 MEDLINE on STN DUPLICATE 10
AU Shihabuddin L S; Palmer T D; Gage F H
TI The search for **neural progenitor cells**: prospects for the therapy of neurodegenerative disease.
SO Molecular medicine today, (1999 Nov) 5 (11) 474-80. Ref: 35
Journal code: 9508560. ISSN: 1357-4310.

L6 ANSWER 40 OF 47 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
AU Muir J K; Raghupathi R; Saatman K E; Wilson C A; Lee V M Y; Trojanowski J Q; Philips M F; McIntosh T K (Reprint)
TI Terminally differentiated human neurons survive and integrate following transplantation into the traumatically injured rat brain
SO JOURNAL OF NEUROTRAUMA, (MAY 1999) Vol. 16, No. 5, pp. 403-414.
Publisher: MARY ANN LIEBERT INC PUBL, 2 MADISON AVENUE, LARCHMONT, NY 10538.
ISSN: 0897-7151.

L6 ANSWER 41 OF 47 CAPLUS COPYRIGHT 2004 ACS on STN
AU Qureshi, Nazer H.; Chiocca, E. Antonio
TI A review of gene therapy for the treatment of central nervous system tumors
SO Critical Reviews in Oncogenesis (1999), 10(4), 261-274
CODEN: CRONEI; ISSN: 0893-9675

L6 ANSWER 42 OF 47 CAPLUS COPYRIGHT 2004 ACS on STN
AU Milward, Elizabeth A.; Lundberg, Cathryn G.; Ge, Bin; Lipsitz, David; Zhao, Ming; Duncan, Ian D.

TI Isolation and transplantation of multipotential populations of epidermal growth factor-responsive, **neural progenitor cells** from the canine brain
SO Journal of Neuroscience Research (1997), 50(5), 862-871
CODEN: JNREDK; ISSN: 0360-4012

L6 ANSWER 43 OF 47 CAPLUS COPYRIGHT 2004 ACS on STN
AU Sakuragawa, Norio
TI Hereditary metabolic disease with convulsion and therapeutic approach
SO Igaku no Ayumi (1997), 183(1), 43-47
CODEN: IGAYAY; ISSN: 0039-2359

L6 ANSWER 44 OF 47 CAPLUS COPYRIGHT 2004 ACS on STN
IN Cauley, Keith; Kukekov, Valery G.
TI Human oligodendroglial progenitor cell line, exogenous gene expression, and use for gene therapy or neuropharmaceutical drug discovery
SO PCT Int. Appl., 41 pp.
CODEN: PIXXD2

L6 ANSWER 45 OF 47 MEDLINE on STN
AU Kahn A; Haase G; Akli S; Guidotti J E
TI [Gene therapy of neurological diseases].
Therapie genique des maladies neurologiques.
SO Comptes rendus des seances de la Societe de biologie et de ses filiales, (1996) 190 (1) 9-11.
Journal code: 7505439. ISSN: 0037-9026.

L6 ANSWER 46 OF 47 MEDLINE on STN DUPLICATE 11
AU Kesari S; Randazzo B P; Valyi-Nagy T; Huang Q S; Brown S M; MacLean A R; Lee V M; Trojanowski J Q; Fraser N W
TI Therapy of experimental human brain tumors using a neuroattenuated herpes simplex virus mutant.
SO Laboratory investigation; a journal of technical methods and pathology, (1995 Nov) 73 (5) 636-48.
Journal code: 0376617. ISSN: 0023-6837.

L6 ANSWER 47 OF 47 MEDLINE on STN DUPLICATE 12
AU Snyder E Y; Taylor R M; Wolfe J H
TI **Neural progenitor cell** engraftment corrects lysosomal storage throughout the MPS VII mouse brain.
SO Nature, (1995 Mar 23) 374 (6520) 367-70.
Journal code: 0410462. ISSN: 0028-0836.

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=> s hib5(w)cell
L1          108 HIB5(W) CELL

=> dup rem l1
PROCESSING COMPLETED FOR L1
L2           53 DUP REM L1 (55 DUPLICATES REMOVED)

=> d bib ab 1-10 l2

L2   ANSWER 1 OF 53     MEDLINE on STN                    DUPLICATE 1
AN   2004023089     MEDLINE
DN   PubMed ID: 14720513
TI   Induction of neurites by the regulatory domains of PKCdelta and epsilon is
     counteracted by PKC catalytic activity and by the RhoA pathway.
AU   Ling Mia; Troller Ulrika; Zeidman Ruth; Lundberg Cecilia; Larsson Christer
CS   Department of Laboratory Medicine, Molecular Medicine, Lund University,
     Malmo University Hospital, 205 02 Malmo, Sweden.
SO   Experimental cell research, (2004 Jan 1) 292 (1) 135-50.
     Journal code: 0373226. ISSN: 0014-4827.
CY   United States
DT   Journal; Article; (JOURNAL ARTICLE)
LA   English
FS   Priority Journals
EM   200402
ED   Entered STN: 20040115
     Last Updated on STN: 20040302
     Entered Medline: 20040227
AB   We have shown that protein kinase C (PKC) epsilon, independently of its
     kinase activity, via its regulatory domain (RD), induces neurites in
     neuroblastoma cells. This study was designed to evaluate whether the same
     effect is obtained in nonmalignant neural cells and to dissect mechanisms
     mediating the effect. Overexpression of PKCepsilon resulted in neurite
     induction in two immortalised neural cell lines (HiB5 and RN33B). Phorbol
     ester potentiated neurite outgrowth from PKCepsilon-overexpressing cells
     and led to neurite induction in cells overexpressing PKCdelta. The
     effects were potentiated by blocking the PKC catalytic activity with
     GF109203X. Furthermore, kinase-inactive PKCdelta induced more neurites
     than the wild-type isoform. The isolated regulatory domains of novel PKC
     isoforms also induced neurites. Experiments with PKCdelta-overexpressing
     HiB5 cells demonstrated that phorbol ester, even in the
     presence of a PKC inhibitor, led to a decrease in stress fibres,
     indicating an inactivation of RhoA. Active RhoA blocked PKC-induced
     neurite outgrowth, and inhibition of the RhoA effector ROCK led to neurite
     outgrowth. This demonstrates that neurite induction by the regulatory
     domain of PKCdelta can be counteracted by PKCdelta kinase activity, that
     PKC-induced neurite outgrowth is accompanied by stress fibre dismantling
     indicating an inactivation of RhoA, and that the RhoA pathway suppresses
     PKC-mediated neurite outgrowth.

L2   ANSWER 2 OF 53  BIOSIS  COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN   2004:104585  BIOSIS
DN   PREV200400096191
TI   Low-level tyrosine hydroxylase (TH) expression allows for the generation
     of stable TH+ cell lines of human neural stem cells.
AU   Liste, Isabel; Navarro, Beatriz; Johansen, Jens; Bueno, Carlos; Villa,
     Ana; Johansen, Teit E.; Martinez-Serrano, Alberto [Reprint Author]
CS   Center of Molecular Biology Severo Ochoa, Department of Molecular Biology,
     Autonomous University of Madrid, Campus Cantoblanco, Madrid, 28049, Spain
     amserrano@cbm.uam.es
SO   Human Gene Therapy, (January 2004) Vol. 15, No. 1, pp. 13-20. print.
     ISSN: 1043-0342 (ISSN print).
DT   Article
LA   English
```

ED Entered STN: 18 Feb 2004
Last Updated on STN: 18 Feb 2004
AB Genetic engineering of neurotransmitter metabolic routes is important for the development of neurotransmitter-producing cells for the ex vivo gene therapy of many CNS diseases. Human neural stem cells (hNSCs) are excellent candidates to serve this role, but, for the case of Parkinson's disease, the cells do not normally express the rate-limiting dopamine (DA) synthesis enzyme tyrosine hydroxylase (TH), and are not equipped with the detoxifying mechanisms needed to prevent the neurotoxicity associated with the DA phenotype. In this study we have examined the capacity of hNSCs for ectopic expression of human TH. High-level TH expression (from viral promoters) leads to growth arrest and hNSC death (associated with an increase in p53 expression and nuclear fragmentation), which can be counteracted by treatment with a pan-caspase inhibitor. As a consequence, stable TH-expressing hNSC sublines could not be derived using viral promoters. In contrast, moderate TH expression (from a human housekeeping promoter, polyubiquitin gene), allows for stable TH+ subclone derivation, seemingly originating from low-expressing cells. Our results are thus compatible with the view that stable TH-expressing hNSC lines can be generated if TH expression levels are kept at a moderate level, and that the goal normally set of aiming at high-level TH expression may need to be reconsidered. These results may be relevant for the generation of THJDA-producing human neural cells for in vitro and neurotransplantation research in Parkinson's disease.

L2 ANSWER 3 OF 53 MEDLINE on STN DUPLICATE 2
AN 2003580075 IN-PROCESS
DN PubMed ID: 14661183
TI Increased in vitro and in vivo transgene expression levels mediated through cis-acting elements.
AU Johansen Jens; Tornoe Jens; Moller Arne; Johansen Teit E
CS NsGene A/S, Ballerup, Denmark.. jjha@lundbeck.com
SO journal of gene medicine, (2003 Dec) 5 (12) 1080-9.
Journal code: 9815764. ISSN: 1099-498X.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS IN-PROCESS; NONINDEXED; Priority Journals
ED Entered STN: 20031216
Last Updated on STN: 20040107
AB BACKGROUND: Gene therapy for neurodegenerative diseases depends critically on the vector system to direct sustained and stable expression of the transgene. It is, however, a commonly observed phenomenon that transgene expression from currently available vectors is down-regulated following ex vivo gene transfer to the central nervous system (CNS). In an attempt to circumvent this problem, we have systematically evaluated the potential of different cis-acting elements to increase and stabilize transgene expression in vitro and after grafting of engineered cell lines to the CNS. METHODS: Plasmid vector constructs incorporating Woodchuck hepatitis post-transcriptional regulatory element (WPRE), cHS4 insulator elements and/or the translational enhancer element SP163 were produced. Stable, polyclonal cultures of **HiB5 cells** were generated by transfection with reporter constructs, and in vitro transgene mRNA and protein levels were determined. Finally, HiB5 clones engineered to express the enhanced green fluorescent protein (EGFP) were grafted to the rat striatum and expression levels were evaluated. RESULTS: Inserting the WPRE element downstream of the open reading frame (ORF) of a reporter gene and flanking the transcriptional unit with cHS4 insulator elements significantly increased protein and mRNA expression levels. Surprisingly, the SP163 element, previously reported to be a translational enhancer, apparently did not promote any translational enhancing activity. Furthermore, the SP163 element exerted a negative effect on transcription. The ability of cHS4 and WPRE elements to stabilize in vivo transgene expression was demonstrated by transplantation of HiB5 clones containing

expression constructs into the rat striatum. CONCLUSION: The data suggest that incorporating cis-acting elements in gene therapy vectors may result in improvements to currently available therapeutic vectors.

L2 ANSWER 4 OF 53 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2004:86901 BIOSIS
DN PREV200400089734
TI The basic helix-loop-helix transcription factor TAL1/SCL inhibits the expression of the p16INK4A and pTalpha genes.
AU Hansson, Anders; Manetopoulos, Christina; Jonsson, Jan-Ingvar; Axelson, Hakan [Reprint Author]
CS Department of Laboratory Medicine, Division of Molecular Medicine, Lund University, University Hospital MAS, SE-205 02, Malmo, Sweden
hakan.axelson@molmed.mas.lu.se
SO Biochemical and Biophysical Research Communications, (December 26 2003) Vol. 312, No. 4, pp. 1073-1081. print.
CODEN: BBRCA9. ISSN: 0006-291X.
DT Article
LA English
ED Entered STN: 11 Feb 2004
Last Updated on STN: 11 Feb 2004
AB The Tal1 gene (also called Scl or TCL5) encodes a basic helix-loop-helix transcription factor required for hematopoiesis and vasculogenesis. Additionally, aberrant transcriptional activation of the Tall gene is a frequent event in human T cell acute lymphoblastic leukemia (T-ALL). T cell specific expression of TAL1 in mice induces aggressive T cell malignancies, demonstrating the oncogenic potential of TAL1. Yet, the underlying mechanisms of TAL1 induced tumorigenesis are poorly understood. By inhibiting E protein mediated transcription of the pTalpha gene, TAL1 can interfere with the T cell differentiation program. In addition, several studies suggest that TAL1 expression might also enhance proliferation rate. We report here that TAL1 can bind the E boxes in both the p16 and the pTalpha promoters, and functionally suppress the activity of both promoters. These results indicate that TAL1 can affect both T cell proliferation and differentiation. Moreover, we show that overexpression of TAL1 in hematopoietic progenitor cells promotes cell cycle division.

L2 ANSWER 5 OF 53 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2003:477660 BIOSIS
DN PREV200300477660
TI The Lim-only protein LMO4 modulates the transcriptional activity of HEN1.
AU Manetopoulos, Christina; Hansson, Anders; Karlsson, Jenny; Jonsson, Jan-Ingvar; Axelson, Hakan [Reprint Author]
CS Department of Laboratory Medicine, Division of Molecular Medicine, Lund University, University Hospital MAS, Malmo, S-205 02, Sweden
hakan.axelson@molmed.mas.lu.se
SO Biochemical and Biophysical Research Communications, (August 8 2003) Vol. 307, No. 4, pp. 891-899. print.
CODEN: BBRCA9. ISSN: 0006-291X.
DT Article
LA English
ED Entered STN: 15 Oct 2003
Last Updated on STN: 15 Oct 2003
AB The basic helix-loop-helix protein HEN1 and the LIM-only proteins LMO2 and LMO4 are expressed in neuronal cells. HEN1 was cloned by virtue of its homology to TAL1, a bHLH protein important for early hematopoiesis. Since it has been shown that TAL1 forms complex with LMO proteins in erythroid and leukemic cells we investigated the capacity of HEN1 to form complex with LMO2 and LMO4. By mammalian two-hybrid analysis, we show that HEN1 interacts with both LMO2 and LMO4. To characterize the transcriptional capacity of HEN1 alone or together with LMO2 and LMO4, we performed reporter gene assays. In comparison with the ubiquitously expressed bHLH

protein E47, HEN1 is a very modest transcriptional activator and titration experiments indicate that HEN1, like TAL1, represses E47 mediated transcriptional activation. Furthermore, LMO4 but not LMO2 was able to augment this effect. Overexpression of HEN1 in hippocampal precursor cells resulted in neurite extension, which could be prevented by LMO4. Taken together, these results indicate that LMO proteins can modulate the transcriptional activity of HEN1.

L2 ANSWER 6 OF 53 MEDLINE on STN DUPLICATE 3
AN 2004001101 IN-PROCESS
DN PubMed ID: 14696671
TI Protective effect of shenqi-wan against H2O2-induced apoptosis in hippocampal neuronal cells.
AU Shin Hyun-Taeg; Chung Seok-Hee; Lee Jong-Soo; Kim Sung-Soo; Shin Hyun-Dae; Jang Mi-Hyeon; Shin Min-Chul; Bahn Geon-Ho; Paik Eun-Kyung; Park Jae-Hyung; Kim Chang-Ju
CS Department of Oriental Rehabilitation Medicine, College of Oriental Medicine Kyung Hee University, Seoul, Korea.
SO American journal of Chinese medicine, (2003) 31 (5) 675-86.
Journal code: 7901431. ISSN: 0192-415X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS IN-PROCESS; NONINDEXED; Priority Journals
ED Entered STN: 20040106
Last Updated on STN: 20040106
AB The present study investigated whether Shenqi-wan possesses a protective effect against hydrogen peroxide (H2O2)-induced apoptosis of the hippocampal cell line HiB5. Through morphological and biochemical analyses, it was demonstrated that **HiB5 cells** treated with H2O2 exhibited several apoptotic features, while cells pre-treated with Shenqi-wan prior to H2O2 exposure showed a decrease in the occurrence of apoptosis. In addition, a patch clamp study revealed that Shenqi-wan inhibited profoundly N-methyl-D-aspartic acid (NMDA) receptor-activated ion current in acutely dissociated hippocampal CA1 neurons. These results suggest that Shenqi-wan may exert its protective effect against H2O2-induced apoptosis via inhibition of NMDA receptors in hippocampal neuronal cells.

L2 ANSWER 7 OF 53 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2004:173400 BIOSIS
DN PREV200400174684
TI Nonspecific association of 2',3'-cyclic nucleotide 3'-phosphodiesterase with the rat forebrain postsynaptic density fraction.
AU Cho, Sun-Jung; Jung, Jae Seob; Shin, Seung Chul; Jin, Ingnyol; Ko, Bok Hyun; Kwon, Yunhee Kim; Suh-Kim, Haeyoung; Moon, Il Soo [Reprint Author]
CS Department of Anatomy, College of Medicine, Medical Institute of Dongguk University, Gyeongju, 780-714, South Korea
moonis@dongguk.ac.kr
SO Experimental & Molecular Medicine, (December 31 2003) Vol. 35, No. 6, pp. 486-493. print.
ISSN: 1226-3613.
DT Article
LA English
ED Entered STN: 31 Mar 2004
Last Updated on STN: 31 Mar 2004
AB The 2',3'-cyclic nucleotide 3'-phosphodiesterase (CNP), a protein of unknown function in vivo, is abundantly expressed in myelinating glia in two isoforms, CNP1 and CNP2. In this study, immunoblot analysis showed that CNP1 is the major isoform in adult forebrain, and that both isoforms are included in the postsynaptic density (PSD) fraction and tyrosine-phosphorylated at the basal level. However, subcellular distribution and detergent extraction data showed that CNP is nonspecifically associated with the PSD fraction. Immunocytochemistry

revealed that CNP is detected, in a weak but punctate pattern, in dissociated rat hippocampal neurons of 3 days to 2 weeks in vitro. The CNP-positive punctae were distributed throughout soma and dendrites, and distinct from PSD95-positive ones. Immunoblot analysis indicated that CNP is also expressed in neuronal stern cell lines, HiB5 and F11. Interestingly, in addition to the known two isoforms, a new CNP isoform of MW 45 kDa was expressed in these cell lines and was the major type of isoform in F11 cells. Taken together, our data suggest that CNP is expressed in the early stage of in vitro development and nonspecifically included in the adult rat PSD fraction.

L2 ANSWER 8 OF 53 CAPLUS COPYRIGHT 2004 ACS on STN DUPLICATE 4
AN 2003:483922 CAPLUS
DN 139:114740
TI The effect of brain-derived neurotrophic factor on neuritogenesis and synaptic plasticity in Aplysia neurons and the hippocampal cell line HiB5
AU Lee, Seung-Hee; Han, Jin-Hee; Choi, Jung-Hwan; Huh, Eun-Young; Kwon, Yunhee Kim; Kaang, Bong-Kiun
CS National Research Laboratory of Neurobiology, Institute of Molecular Biology and Genetics, School of Biological Sciences, Seoul National University, Seoul, 151-742, S. Korea
SO Molecules and Cells (2003), 15(2), 233-239
CODEN: MOCEEK; ISSN: 1016-8478
PB Korean Society for Molecular and Cellular Biology
DT Journal
LA English
AB Brain-derived neurotrophic factor (BDNF) plays a key role in the differentiation and neuritogenesis of developing neurons, and in the synaptic plasticity of mature neurons, in the mammalian nervous system. BDNF binds to the receptor tyrosine kinase TrkB and transmits neurotrophic signals by activating neuron-specific tyrosine phosphorylation pathways. However, the neurotrophic function of BDNF in Aplysia neurons is poorly understood. The authors examined the specific effect of BDNF on neurite outgrowth and synaptic plasticity in cultured Aplysia neurons and a multipotent rat hippocampal stem cell line (HiB5). The authors' study indicates that mammalian BDNF has no significant effect on the neuritogenesis, neurotransmitter release, excitability, and synaptic plasticity of cultured Aplysia neurons in the authors' exptl. conditions. In contrast, BDNF in combination with platelet-derived growth factor (PDGF) increases the length of the neurites and the number of spine-like structures in cells of HiB5.
RE.CNT 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 9 OF 53 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2004:205324 BIOSIS
DN PREV200400205851
TI Upregulation of beta - Pix expression in migrating neural stem cells in the rat brain.
AU Huh, E. [Reprint Author]; Yoon, J. [Reprint Author]; Lim, J. [Reprint Author]; Park, D.; Chung, J.; Kwon, Y. K. [Reprint Author]
CS Dept. of Biol., Kyunghee Univ, Seoul, South Korea
SO Society for Neuroscience Abstract Viewer and Itinerary Planner, (2003) Vol. 2003, pp. Abstract No. 885.9. http://sfn.scholarone.com. e-file. Meeting Info.: 33rd Annual Meeting of the Society of Neuroscience. New Orleans, LA, USA. November 08-12, 2003. Society of Neuroscience.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 14 Apr 2004
Last Updated on STN: 14 Apr 2004
AB The small GTPases of the Rho family play roles in transducing extracellular stimuli into distinct responses including cell motility, adhesion, cell division and phagocytosis. The GTPases cycle between

GTP-bound and GDP-bound forms and their activation requires the action of guanine nucleotide exchange factors (GEFs) to promote the conversion of the GDP to the GTP state. beta-Pix is p21-activated kinase (PAK)-interacting exchange factor (PIX). It encodes a guanine nucleotide exchange factor for Rho guanosisne triphosphatases (Rac/Cdc42) and known to regulate actin cytoskeleton reorganization, cooperating with Rac, PAK and GIT. It contains SH3 (Src homology 3), DH(Dbl homology), PH(Pleckstrin homology) , PXXP, Insert region domain. **HiB5 cells** are neuronal stem cells derived from embryonic day16 (E16) rat hippocampus.PDGF treated **HiB5 cells** migrate along hippocampal alveus by 4weeks when transplanted into the rat hippocampus. We investigated whether the migrating neural stem cells express beta-pix. Isoforms of beta-Pix were detected in **HiB5 cells** using antibody raised against beta-Pix insert domain. PDGF treated **HiB5 cells** migrating along hippocampal alveus increasd expression of beta-Pix and paxillin than PDGF untreated **HiB5 cells**. The result of this study suggest that PDGF enhances migration of transplanted **HiB5 cells** and beta-Pix regulates migration and differentiation.

L2 ANSWER 10 OF 53 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2002:31496 CAPLUS
DN 136:65968
TI A lamotrigine-sensitive sodium channel RNAv1.5a from rat astrocytes
IN Korsgaard, Mads P. G.
PA Neurosearch A/s, Den.
SO PCT Int. Appl., 12 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2002002608	A2	20020110	WO 2001-DK414	20010614
	WO 2002002608	A3	20020510		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	EP 1301596	A2	20030416	EP 2001-940247	20010614
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
PRAI	DK 2000-1049	A	20000705		
	WO 2001-DK414	W	20010614		

AB The present invention relates to a novel voltage gated sodium channel located in the brain, a cDNA encoding it, vectors and host cells containing the same, transgenic non-human animal capable of expressing the sodium channel, and methods of screening for modulators of the channel such as modulators for use in the treatment of seizures, and conditions related to the limbic system and limbic regions including limbic seizures. The sodium channel, a sequence homolog of the rat RNAv1.5 channel was identified by RT-PCR during an anal. of sodium channel gene expression in **HiB5 cells**. The protein was more sensitive to inhibition by lamotrigine than the RNAv1.5 channel, suggesting that it may be a target for the treatment of epilepsy.

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=> d his

     (FILE 'HOME' ENTERED AT 15:02:07 ON 14 MAY 2004)

     FILE 'MEDLINE, CAPLUS, BIOSIS, SCISEARCH' ENTERED AT 15:02:23 ON 14 MAY
     2004
L1          3012 S (NEURONAL OR NEURAL)(3A)PROGENITOR(W)CELL
L2         66482 S (TREAT? OR PROTECT?)(6A)(NEURAL(W)TISSUE OR BRAIN OR CNS OR C
L3            73 S L1 AND L2
L4            13 S L1(9A)L2
L5            11 DUP REM L4 (2 DUPLICATES REMOVED)
L6            47 DUP REM L3 (26 DUPLICATES REMOVED)

=> d bib ab 1-11 l5

L5   ANSWER 1 OF 11  CAPLUS  COPYRIGHT 2004 ACS on STN
AN   2003:376662  CAPLUS
DN   138:379246
TI   The functional role and potential therapeutic use of Reelin, Gas6 and
     Protein S in relation to adult neural stem or progenitor cells
IN   Bertilsson, Goran; Frisen, Jonas; Falk, Anna; Heidrich, Jessica;
     Hellstrom, Kristina; Kortesmaa, Jarkko; Lindquist, Per; Lundh, Hanna;
     McGuire, Jaccqueline; Mercer, Alex; Patrone, Cesare; Ronnholm, Harriet;
     Wikstrom, Lilian; Zachrisson, Olof
PA   Neuronova AB, Swed.
SO   PCT Int. Appl., 112 pp.
     CODEN: PIXXD2
DT   Patent
LA   English
FAN.CNT 1
     PATENT NO.        KIND  DATE           APPLICATION NO.  DATE
     ---------------   ----  --------       ---------------  --------
PI   WO 2003039575      A2   20030515       WO 2002-GB5078   20021111
     WO 2003039575      A3   20031016
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
             CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
             GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
             LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH,
             PL, PT, RO, RU, SC, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT,
             TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ,
             MD, RU, TJ, TM
         RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG,
             CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL,
             PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR,
             NE, SN, TD, TG
     US 2003165485      A1   20030904       US 2002-291171   20021108
PRAI US 2001-344725P    P    20011109
     US 2001-345064P    P    20011109
     US 2002-393263P    P    20020702
     US 2002-394397P    P    20020708
AB   The invention relates generally to methods of influencing central nervous
     system cells to produce progeny useful in the treatment of CNS disorders.
     More specifically, the invention includes methods of exposing a patient
     suffering from such a disorder to reagent that modulates the
     proliferation, migration, differentiation and survival of central nervous
     system cells via Reelin, Gas6 or Protein S signaling.  These methods are
     useful for reducing at least one symptom of the disorder.

L5   ANSWER 2 OF 11  SCISEARCH  COPYRIGHT 2004 THOMSON ISI on STN
AN   2003:554377  SCISEARCH
GA   The Genuine Article (R) Number: 692TP
TI   Nitric oxide acts in a positive feedback loop with BDNF to regulate neural
     progenitor cell proliferation and differentiation in the mammalian brain
```

AU Cheng A W; Wang S Q; Cai J L; Rao M S; Mattson M P (Reprint)
CS NIA, Gerontol Res Ctr, Neurosci Lab, 5600 Nathan Shock Dr, Baltimore, MD 21224 USA (Reprint); NIA, Gerontol Res Ctr, Neurosci Lab, Baltimore, MD 21224 USA
CYA USA
SO DEVELOPMENTAL BIOLOGY, (15 JUN 2003) Vol. 258, No. 2, pp. 319-333.
Publisher: ACADEMIC PRESS INC ELSEVIER SCIENCE, 525 B ST, STE 1900, SAN DIEGO, CA 92101-4495 USA.
ISSN: 0012-1606.
DT Article; Journal
LA English
REC Reference Count: 62
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Nitric oxide (NO) is believed to act as an intercellular signal that regulates synaptic plasticity in mature neurons. We now report that NO also regulates the proliferation and differentiation of mouse **brain neural progenitor cells** (NPCs). **Treatment** of dissociated mouse cortical neuroepithelial cluster cell cultures with the NO synthase inhibitor L-NAME or the NO scavenger hemoglobin increased cell proliferation and decreased differentiation of the NPCs into neurons, whereas the NO donor sodium nitroprusside inhibited NPC proliferation and increased neuronal differentiation. Brain-derived neurotrophic factor (BDNF) reduced NPC proliferation and increased the expression of neuronal NO synthase (nNOS) in differentiating neurons. The stimulatory effect of BDNF on neuronal differentation of NPC was blocked by L-NAME and hemoglobin, suggesting that NO produced by the latter cells inhibited proliferation and induced neuronal differentiation of neighboring NPCs. A similar role for NO in regulating the switch of neural stem cells from proliferation to differentiation in the adult brain is suggested by data showing that NO synthase inhibition enhances NPC proliferation and inhibits neuronal differentiation in the subventricular zone of adult mice. These findings identify NO as a paracrine messenger stimulated by neurotrophin signaling in newly generated neurons to control the proliferation and differentiation of NPC, a novel mechanism for the regulation of developmental and adult neurogenesis.

L5 ANSWER 3 OF 11 MEDLINE on STN DUPLICATE 1
AN 2003287176 MEDLINE
DN PubMed ID: 12814945
TI Differentiation and morphological integration of neural progenitor cells transplanted into the developing mammalian eye.
AU Sakaguchi D S; Van Hoffelen S J; Young M J
CS Department of Zoology and Genetics, Iowa State University, Ames, Iowa 50011, USA.. dssakagu@iastate.edu
NC 09595
SO Annals of the New York Academy of Sciences, (2003 May) 995 127-39. Ref: 26
Journal code: 7506858. ISSN: 0077-8923.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 200307
ED Entered STN: 20030620
Last Updated on STN: 20030723
Entered Medline: 20030722
AB Transplantation of neural stem/progenitor cells has been proposed as a novel approach for the replacement and repair of damaged CNS tissues. We have evaluated the influence of the host cellular microenvironment upon the survival, differentiation, and integration of neural progenitor cells

transplanted into the CNS. Using this approach, we have investigated the fate of neural progenitor cells in vivo following transplantation into the developing mammalian eye. Murine brain progenitor cells (mBPCs) isolated from neonatal mice expressing the green fluorescent protein (GFP) transgene were transplanted into the eyes of Brazilian opossums (Monodelphis domestica). Monodelphis pups are born in an extremely immature, fetal-like state. The eyes of neonatal pups provide a fetal-like environment in which to study cellular interactions between host tissues and transplanted neural progenitor cells. mBPCs were transplanted by intraocular injection in hosts ranging in age from 5 days postnatal to adult. The transplanted cells were easily identified because of their GFP fluorescence. Extensive survival, differentiation, and morphological integration of mBPCs within the host tissue was observed. We found that the younger retinas provided a more supportive environment for the morphological integration of the transplanted mBPCs. Cells with morphologies characteristic of specific retinal cell types were observed. Moreover, some transplanted mBPCs were labeled with antibodies characteristic of specific neural/retinal phenotypes. These results suggest that the host environment strongly influences progenitor cell differentiation and that transplantation of **neural progenitor cells** may be a useful approach aimed at **treating** degeneration and pathology of the **CNS**.

L5 ANSWER 4 OF 11 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2004:194632 BIOSIS
DN PREV200400195191
TI Transplantation of neural progenitor cells and fibroblasts into motor cortex and striatum of naive rats: Survival, migration, differentiation, and functional outcome.
AU Price, R. O. [Reprint Author]; Pedatella, K. E. [Reprint Author]; Schallert, T. J.; Palmer, T. D. [Reprint Author]
CS Neurosurgery, Stanford Univ., Stanford, CA, USA
SO Society for Neuroscience Abstract Viewer and Itinerary Planner, (2003) Vol. 2003, pp. Abstract No. 150.10. http://sfn.scholarone.com. e-file. Meeting Info.: 33rd Annual Meeting of the Society of Neuroscience. New Orleans, LA, USA. November 08-12, 2003. Society of Neuroscience.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 14 Apr 2004
Last Updated on STN: 14 Apr 2004
AB **Neural progenitor cells** (NPCs) are being investigated as a **treatment** modality for various **brain** pathologies but the precise role NPCs play in functional recovery remains unclear. The goal of this study is to examine cellular behavior and the effects of cell transplantation on motor function in the healthy brain. NPCs were compared with skin fibroblasts (1X105 cells/mul) or saline. Cells were pre-labeled with bromodeoxyuridine (BrdU) and stereotaxically injected into the putamen and primary motor cortex of adult rats. One cell type was placed in the left hemisphere and saline, or the opposite cell type, placed in the right hemisphere. Sensory and motor behavior in the vertical exploration, ledged-tapered beam, and adhesive-removal paradigms were assessed for 28 days post-transplant. The brains were removed and evaluated for cell survival, migration, and fate. Behavioral data suggest that NPCs produce an asymmetry in forelimb use (increase in use of the forelimb opposite to the NPC transplants) when saline-only was transplanted in the contralateral hemisphere. Fibroblasts transplanted contralateral to the NPCs appear to negate the asymmetry. Histologically, NPCs migrate extensively and differentiate variably depending upon the distance from the graft and location within the brain. In contrast, fibroblasts have reduced survival and do not migrate. In these pilot studies, it appears that NPCs and fibroblast transplants can influence behavior, but the effects on motor function may be independent of cell source, extent of migration, or phenotype of surviving cells.

L5 ANSWER 5 OF 11 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
AN 2002:26101 SCISEARCH
GA The Genuine Article (R) Number: 505CY
TI Stem cells in neurodevelopment and plasticity
AU Vaccarino F M (Reprint); Ganat Y; Zhang Y C; Zheng W
CS Yale Univ, Ctr Child Study, 230 S Frontage Rd, New Haven, CT 06520 USA (Reprint); Yale Univ, Ctr Child Study, New Haven, CT 06520 USA; Yale Univ, Neurobiol Sect, New Haven, CT 06520 USA
CYA USA
SO NEUROPSYCHOPHARMACOLOGY, (DEC 2001) Vol. 25, No. 6, pp. 805-815.
Publisher: ELSEVIER SCIENCE INC, 655 AVENUE OF THE AMERICAS, NEW YORK, NY 10010 USA.
ISSN: 0893-133X.
DT General Review; Journal
LA English
REC Reference Count: 100
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB The processes of stem cell proliferation and differentiation during embryogenesis are governed by transcription factors that regulate the regional differentiation of the central nervous system (CNS). Do neural "stem" cells persisting in the postnatal CNS disobey this sequence of events? The division of neural progenitor cells is promoted by basic Fibroblast Growth Factor Fgf2 or Epidermal Growth Factor Egf. However, while the intraventricular administration of FgF2 during embryogenesis increases the generation of cortical pyramidal neurons, the same **treatment** in the adult **CNS** produces interneurons of the olfactory bulb. The competence of **neural progenitor cells** to respond to Fgf is dictated by nuclear transcription factors that constrain neuronal fates through time. Developmentally regulated transcriptional programs arc regulated by cell interactions, as dividing cells check their molecular signature against that of their environment. Thus, cell surface interactions account for competitive phenomena among pools of cells, including the inhibitory effect of neurons on the division of their progenitors, and may also explain the "permissive" effects of non-CNS environments. The challenge remains to understand the genetic programs that control the fate of progenitor cells within the postnatal CNS and their regulation by stress, apoptosis and environmental perturbations. These programs are likely to be similar to gene cascades that control proliferation, differentiation and migration of progenitor cells at earlier stages of development.
[Neuropsychopharmacology 25:805-815,2001] (C) 2001 American College of Neuropsychopharmacology. Published by Elsevier Science Inc.

L5 ANSWER 6 OF 11 SCISEARCH COPYRIGHT 2004 THOMSON ISI on STN
AN 2001:976110 SCISEARCH
GA The Genuine Article (R) Number: 498QK
TI Telomerase protects developing neurons against DNA damage-induced cell death
AU Lu C B; Fu W M; Mattson M P (Reprint)
CS NIA, Gerontol Res Ctr, Neurosci Lab, 5600 Nathan Shock Dr, Baltimore, MD 21224 USA (Reprint); NIA, Gerontol Res Ctr, Neurosci Lab, Baltimore, MD 21224 USA; Johns Hopkins Univ, Sch Med, Dept Neurosci, Baltimore, MD 21205 USA
CYA USA
SO DEVELOPMENTAL BRAIN RESEARCH, (26 NOV 2001) Vol. 131, No. 1-2, pp. 167-171.
Publisher: ELSEVIER SCIENCE BV, PO BOX 211, 1000 AE AMSTERDAM, NETHERLANDS.
ISSN: 0165-3806.
DT Article; Journal
LA English
REC Reference Count: 34
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB In mitotic cells, telomerase adds repeats of a DNA sequence (TTAGGG) to the ends of chromosomes (telomeres) thereby maintaining their length and preventing cellular senescence. We recently reported that the catalytic subunit of telomerase (TERT) is expressed in **neuronal progenitor cells** and in early postmitotic neurons in the developing rodent **brain**. We now report that TERT can **protect** cultured PC12 cells and embryonic hippocampal neurons against death induced by DNA damage. Overexpression of TERT in PC12 cells increases their resistance to the topoisomerase inhibitors camptothecin and etoposide. Hippocampal neurons in which TERT levels are decreased using antisense technology exhibit increased vulnerability to the DNA-damaging agents. Emerging findings suggest that DNA damage may trigger the death of neurons during brain development and in neurodegenerative disorders. Our data therefore suggest roles for TERT in modulating such cell deaths. Published by Elsevier Science B.V.

L5 ANSWER 7 OF 11 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2001:258284 CAPLUS
DN 134:364662
TI Regulatory mechanisms for the differentiation of neural stem cells
AU Okano, Hideyuki; Sakakibara, Shin-ichi; Sawamoto, Kazunobu; Nakamura, Yuki; Kaneko, Yukiko; Akamatsu, Wado; Tokunaga, Akinori; Imai, Takao; Miyata, Takaki; Shimazaki, Takuya
CS Department of Neuroanatomy CREST, JST, Osaka University Medical School, Osaka, Japan
SO International Congress Series (2001), 1222(Tissue Engineering for Therapeutic Use 5), 11-19
CODEN: EXMDA4; ISSN: 0531-5131
PB Elsevier Science B.V.
DT Journal
LA English
AB Neural stem cells (NSCs) are self-renewing and multipotential **neural progenitor cells**, which have received considerable attention as potential tools for **treating** the injured or diseased **brain**. To develop therapeutic strategies intended to capitalize upon the plasticity and propagative ability of NSCs, we need both to better understand their biol. and to develop better means for their prospective identification and harvest. Here we report several new findings from our labs. that address the isolation of NSCs, as well as the maintenance and regulatory control of the stem cell phenotype. Specifically, we report (1) the identification of Hes 1 as a neg. regulator of NSC differentiation, and (2) the identification of the Musashi and Hu proteins as selective markers for NSCs and their neuronal daughters, resp.
RE.CNT 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 8 OF 11 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2000:493693 CAPLUS
DN 133:100460
TI A method for introducing nucleic acids into neural stem or progenitor cells via the inherent transport system of the cell
IN Eriksson, Peter; Orwar, Owe
PA A+ Science Invest AB, Swed.
SO PCT Int. Appl., 26 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2000042202	A1	20000720	WO 2000-SE73	20000114

W: AE, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, EE, EE, ES, FI, FI, GB, GD, GE,

GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KR, KZ, LC,
LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL,
PT, RO, RU, SD, SE, SG, SI, SK, SK, SL, TJ, TM, TR, TT, TZ, UA,
UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE,
DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF,
CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

	CA 2359349	AA	20000720	CA 2000-2359349	20000114
	EP 1141340	A1	20011010	EP 2000-902243	20000114
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
	AU 751342	B2	20020815	AU 2000-23372	20000114
	JP 2002534126	T2	20021015	JP 2000-593759	20000114
	NZ 513502	A	20030328	NZ 2000-513502	20000114
PRAI	SE 1999-134	A	19990115		
	WO 2000-SE73	W	20000114		

AB A method for introducing a substance comprising a nucleic acid into a mammalian neural stem cell or progenitor cell, characterized in that said nucleic acid directly interacts with the cell membrane of said cell or a component within said cell membrane whereby the substance comprising said nucleic acid is taken up by the cell via the inherent transport mechanism of the cell, is disclosed. The advantages of the present invention are: (1) it does not rely on the binding of DNA to any soluble receptors or carriers; (2) It allows for the selective labeling of cells, due to the fact that only cells with the inherent transport system are transfected. Also different applications of said method are disclosed.

RE.CNT 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 9 OF 11 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2000:368139 CAPLUS
DN 132:343355
TI Growth hormone-modulating agents and method for treatment of conditions affecting neural stem cells or progenitor cells
IN Eriksson, Peter
PA A+ Science Invest AB, Swed.
SO PCT Int. Appl., 22 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2000030675	A2	20000602	WO 1999-SE2197	19991125
	WO 2000030675	A3	20000817		
	W: AE, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	EP 1135156	A2	20010926	EP 1999-963765	19991125
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, IE, SI, LT, LV, FI, RO				
	JP 2002530351	T2	20020917	JP 2000-583558	19991125
	NZ 512495	A	20031031	NZ 1999-512495	19991125
PRAI	SE 1998-4064	A	19981125		
	WO 1999-SE2197	W	19991125		

AB The invention discloses the use of a substance that, on administration, will lead to increased concns. of growth hormone, e.g. growth hormone, a functionally equivalent analog thereof, or a substance that will increase the

release of endogenous growth hormone, for the production of a medicinal product for treatment of abnormal conditions affecting neural stem cells, progenitor cells and/or cells derived from neural stem cells or progenitor cells, especially conditions affecting the oligodendroglia, astroglia, and/or neuronal cells. In vitro and in vivo methods are disclosed for inducing lineage determination, propagating and/or inducing or maintaining the genesis of neurons, oligodendrocytes, astroglial cells from progenitor cells, stem cells and/or cells derived from said cells by administering to the cells a substance that increases the concentration of growth hormone. Also disclosed is a method of reducing the genesis of oligodendrocytes, neurons, or astroglial cells from progenitor cells or stem cells, wherein a pharmaceutically effective amount of a substance that will lead to a decreased concentration of growth hormone or a functionally equivalent analog thereof is administered to the patient.

L5 ANSWER 10 OF 11 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1997:785588 CAPLUS
DN 128:84693
TI Isolation and transplantation of multipotential populations of epidermal growth factor-responsive, neural progenitor cells from the canine brain
AU Milward, Elizabeth A.; Lundberg, Cathryn G.; Ge, Bin; Lipsitz, David; Zhao, Ming; Duncan, Ian D.
CS Department of Medical Sciences, School of Veterinary Medicine, University of Wisconsin, Madison, Madison, WI, 53706, USA
SO Journal of Neuroscience Research (1997), 50(5), 862-871
CODEN: JNREDK; ISSN: 0360-4012
PB Wiley-Liss, Inc.
DT Journal
LA English
AB Glial cell transplantation into myelin-deficient rodent models has resulted in myelination of axons and restoration of conduction velocity. The shaking (sh) pup canine myelin mutant is a useful model in which to test the ability to repair human myelin diseases, but as in humans, the canine donor supply for allografting is limited. A solution may be provided by self-renewing epidermal growth factor (EGF)-responsive multipotential neural progenitor cell populations ("neurospheres"). Nonadherent spherical clusters, similar in appearance to murine neurospheres, have been obtained from the brain of perinatal wildtype (wt) canine brain and expanded in vitro in the presence of EGF for at least 6 mo. Most of the cells in these clusters express a nestin-related protein. Within 1-2 wk after removal of EGF, cells from the clusters generate neurons, astrocytes, and both oligodendroglial progenitors and oligodendrocytes. Transplantation of lacZ-expressing wt neurospheres into the myelin-deficient (md) rat showed that a proportion of the cells differentiated into oligodendrocytes and produced myelin. In addition, cells from the neurosphere populations survived at least 6 wk after grafting into a 14-day postnatal sh pup recipient and at least 2 wk after grafting into an adult sh pup recipient. Thus, neurospheres provide a new source of allogeneic donor cells for transplantation studies in this mutant.
RE.CNT 48 THERE ARE 48 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 11 OF 11 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1997:699510 CAPLUS
DN 127:329571
TI Hereditary metabolic disease with convulsion and therapeutic approach
AU Sakuragawa, Norio
CS Shinkei Kenkyusho, Kokuritsu Seishin, Shinkei Senta, Kodaira, 187, Japan
SO Igaku no Ayumi (1997), 183(1), 43-47
CODEN: IGAYAY; ISSN: 0039-2359
PB Ishiyaku

DT Journal; General Review
LA Japanese
AB A review, with 22 refs., on mol. basis for hereditary metabolic diseases with epileptic convulsion in children including neonatal convulsion and hereditary metabolic diseases with infantile spasm. Development of **neuronal progenitor cells** and its grafting to the **brain** for the **treatment** are also discussed.

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L6 ANSWER 44 OF 47 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1997:61366 CAPLUS
DN 126:71207
TI Human oligodendroglial progenitor cell line, exogenous gene expression, and use for gene therapy or neuropharmaceutical drug discovery
IN Cauley, Keith; Kukekov, Valery G.
PA Signal Pharmaceuticals, Inc., USA
SO PCT Int. Appl., 41 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9638576	A1	19961205	WO 1996-US8158	19960530
	W: AU, CA, JP				
	RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	US 5830651	A	19981103	US 1995-458890	19950601
	AU 9659590	A1	19961218	AU 1996-59590	19960530
PRAI	US 1995-458890		19950601		
	WO 1996-US8158		19960530		